most important factors in determining whether spinal cord damage occurs from a neck injury is the preexisting space available for the spinal cord and not necessarily the severity of the injury.[16]

The central cord syndrome should be considered in any patient with impaired extremity function after a head or neck injury even though the injury is a minor one and also in patients with impaired function of the extremities even if there is no history of head or neck injury.

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Mycotic Aneurysm Presenting as Fever of Unknown Origin

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FEVER OF UNKNOWN ORIGIN was defined by Petersdorf and Beeson in 1961 as fever present for at least three weeks without its cause being determined after a week in hospital and with reasonable investigations.[1] Mycotic aneurysms as a cause of fever of unknown origin are uncommon and are associated with a high mortality due to catastrophic rupture. Osler in 1885 first coined the term "mycotic aneurysm" to describe a mushroom-shaped aneurysm in a patient with infective endocarditis. It has since been used to describe all infective aneurysms except those due to syphilis. Despite all the latest investigative and therapeutic methods available, the diagnosis and early recognition of mycotic aneurysm remains difficult and the treatment continues to be associated with high mortality and morbidity rates. We report a case that clearly highlights the problems encountered in establishing an early diagnosis of mycotic aortic aneurysm as a cause of fever of unknown origin, especially in an elderly patient with prolonged nonspecific symptoms, an absence of localizing physical signs, and at an age when atherosclerotic aortic aneurysms are common.

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Report of a Case

The patient, a 76-year-old retired farmer who had received a Bjork-Shiley mitral valve replacement in 1971 for rheumatic mitral stenosis, but who was otherwise well, presented to his family physician with a sudden onset of fever, rigors, and right acromioclavicular arthralgia. He was admitted to hospital and after receiving physiotherapy and analgesics, was discharged home on June 5, 1987, with resolution of the arthralgia but still febrile. On June 26, he was readmitted with fever, chills, anorexia, malaise, vague abdominal pains, myalgias, increasing ankle edema, occasional cough without expectoration, and a weight loss of 11 kg (25 lb). He received empiric therapy with gentamicin sulfate and cephalothin sodium given intravenously for two days and was transferred to University Hospital in Saskatoon. There was a history of an allergy to penicillin.

On physical examination on admission, he was slightly obese, fully conscious, and oriented, with an irregular pulse rate of 76 per minute, a blood pressure of 140/98 mm of mercury, respirations 18, and a temperature of 37.2°C (98.6°F). Other findings included bilateral pitting edema of the lower legs, a jugular venous pulse 2 cm above the sternal angle at 45 degrees, and the cardiac apex 3 cm to the left of the midclavicular line in the fifth intercostal space. A pansystolic apical murmur of grade II/VI and an aortic ejection

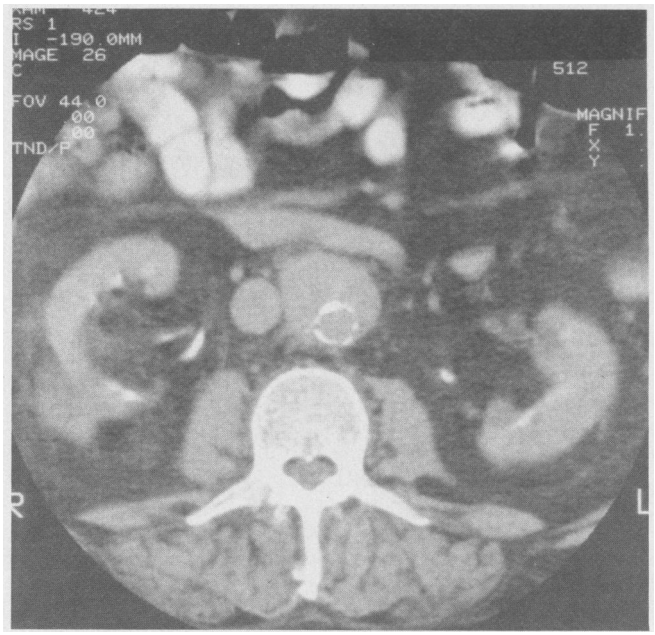

Figure 1.—An axial computed tomographic scan of the abdomen shows ring calcification in the intima of the abdominal aorta with surrounding soft tissue density anteriorly. No gas is present in the soft tissue mass nor is there adjacent bone destruction.

systolic murmur of grade II/VI were audible. All peripheral pulses were palpable. There were no abdominal masses, and the examination revealed no other abnormalities.

Initial laboratory investigations revealed a hemoglobin of 115 grams per liter, a leukocyte count of 24×10^9 per liter, platelet count 322×10^9 per liter, and a notably increased erythrocyte sedimentation rate of 112 mm per hour. The electrolytes, urea, creatinine, and serum aminotransferase values were all normal. A urinalysis revealed 10 erythrocytes and 10 leukocytes per high-power field with a trace of protein. There was cardiomegaly with pulmonary venous hypertension on a chest radiograph. Atrial fibrillation was confirmed by an electrocardiogram.

The patient remained febrile at 38°C to 39°C (100°F to 102°F) and had episodes of chills, anorexia, and episodic ill-defined lower abdominal pain with no new signs on several detailed physical examinations carried out during the hospital stay. An abdominal ultrasonogram done on admission was reported to be normal. The distal abdominal aorta was not visualized. A computed tomographic (CT) scan of the abdomen eight days after admission revealed a 4-cm distal abdominal aortic aneurysm with a 2-cm calcified ring within it (Figure 1). This was thought to be an unusual appearance for an atherosclerotic aneurysm, but the significance was unclear. Ten days after admission, 1 set of blood cultures out of 14 specimens collected showed a growth of *Streptococcus pneumoniae,* and he was treated with a regimen of intravenous gentamicin and vancomycin hydrochloride for a presumptive diagnosis of prosthetic valve endocarditis. This therapy was discontinued after three weeks due to deteriorating renal function, persistent fever ranging from 37.5°C to 38.5°C (99.5°F to 101.3°F), a lack of clinical improvement, and a fall of his hemoglobin level to 70 grams per liter with a leukocyte count of 19.9×10^9 per liter. The alkaline phosphatase level rose from 285 to 627 IU per liter, with the γ-glutamyl transferase value at 363 IU per liter. A subsequent workup during the five-week hospital stay that included fungal blood cultures; a urine culture; echocardiography; a liver biopsy; bone marrow aspiration, biopsy, and culture; serum complement levels; serologic tests for *Brucella* species and Q fever; a urine culture for cytomegalovirus; an upper gastrointestinal series; double-contrast barium enema; endoscopic retrograde cholangiopancreatography; an isotope liver and spleen scan; acid-fast bacilli culture and stains of bone marrow, liver, and sputum; and a temporal artery biopsy did not reveal any significant pathologic process. A total body scan using gallium Ga 67 citrate was done without any abnormality found. The ultrasonogram was repeated (Figure 2) and confirmed the presence of an aneurysm that was eccentric in configuration and suggested a leaking abdominal aneurysm or mycotic aneurysm. During the fifth week an angiogram was done because of the previous ultrasonographic and CT findings. At the time of the angiogram, no right common femoral pulse was palpable. An intra-arterial digital subtraction angiogram (Figure 3) done through the left common femoral artery showed a saccular aneurysm approximately 6 cm in diameter arising eccentrically from the distal abdominal aorta and involving the aortic bifurcation with occlusion of the right common iliac artery. The diagnosis of mycotic abdominal aortic aneurysm was made.

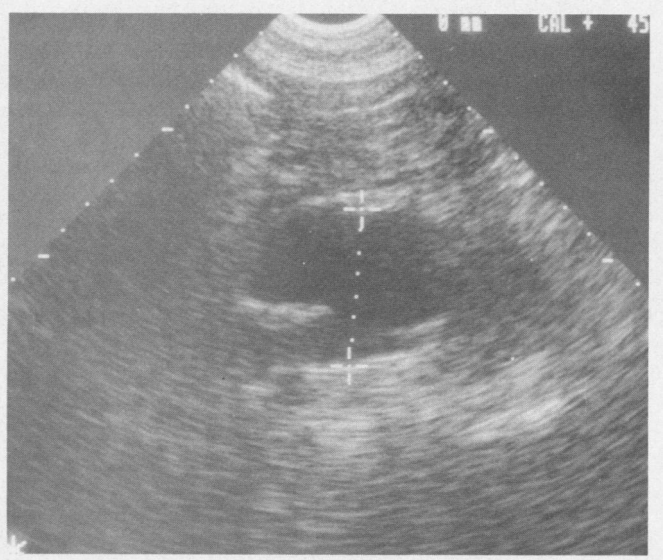

Figure 2.—A sagittal ultrasonogram shows the abdominal aorta posterior to an echo-free mass that communicates with the abdominal aorta at its lower end. The appearance is of a saccular aneurysm rather than the typical fusiform aneurysm of atherosclerosis.

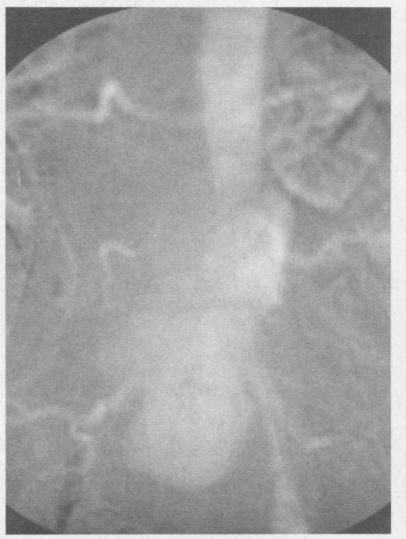

Figure 3.—An intra-arterial digital subtraction aortogram shows a lobulated, 5-cm long contrast collection anterior to the distal abdominal aorta representing the saccular aneurysm with occlusion of the right common iliac artery.

An emergency operation showed an infected abdominal aortic aneurysm below the renal arteries that was ligated and resected. A left axillofemoral bypass and femoral-femoral crossover bypass graft from left to right was carried out to establish peripheral circulation to the lower limbs. The patient made an unremarkable recovery with sustained defervescence beginning one day postoperatively, and he was eventually discharged home with no sequelae.

Because of the risk of recurrent infection at the site of the resected aorta or of seeding of the prosthetic bypass graft, the patient was discharged on a regimen of oral cephalexin, which was to be continued indefinitely.

Discussion

The exact prevalence of aortic mycotic aneurysms is not known. In the preantibiotic era, most were due to infective endocarditis. The risk factors in the postantibiotic era, however, are arterial trauma (29%), depressed immunocompetence (24%), concurrent sepsis (17%), bacterial endocarditis (17%), congenital cardiovascular defects (10%), and primary aortic aneurysm (3%).[2] There is a male preponderance in a ratio of 3:1.[3] The pathogenesis is thought to be due to either septic aortic emboli from infective endocarditis, direct

or lymphatic extension from an adjacent infectious process, or hematogenous spread.[4] The emboli lodge in the vasa vasorum and produce a local arteritis, eventually resulting in necrosis and aneurysm formation.[5] The latest classification of mycotic aneurysms by Patel and Johnson dividing them with regard to the preexisting status of the artery and the source of infection is useful.[5]

In the preantibiotic era, *Streptococcus pyogenes* and *S pneumoniae* were prevalent, whereas in the postantibiotic era, viridans streptococci (30% to 40%), *Staphylococcus aureus* (10% to 25%), enterococci (5% to 15%), gram-negative bacilli (2% to 12%), and *Staphylococcus epidermidis* (1% to 3%) are common infecting organisms. Between 5% and 20% of cases are culture-negative. Cases due to *Salmonella* species have a tendency towards early rupture.[4] Although variable, the clinical presentation[2,6-8] includes fever (75%), back and abdominal pain (33%), abdominal pain and fever (20%), and palpable aneurysm (53%). In most cases, the onset is insidious, and low-grade fever may be present for several months. Because of the nonspecific symptoms and poor awareness of the condition among physicians, 75% rupture preoperatively. Rupture may occur into the peritoneal cavity, pleural cavity, duodenum, esophagus, mediastinum, or the pericardium. Laboratory findings include leukocytosis (65%), positive blood cultures (70%), aortic calcification (47%), and vertebral erosion (18%). Bacteremia is usually continuous and does not resolve with antibiotic treatment alone.

Mycotic aortic aneurysms have been described as "inevitably fatal,"[2] but early diagnosis followed by administering the appropriate antibiotics and timely surgical repair can result in cures of this otherwise lethal condition.[2,9] All patients without surgical treatment die of catastrophic hemorrhage or sepsis. The surgical treatment is aneurysmal resection with aortic interposition grafts or extra-anatomic bypass grafting. The antibiotic therapy should be continued for at least six to eight weeks.

The radiographic features of mycotic aneurysms previously described are erosion of lumbar vertebrae[6,10,11]; abnormal gas collections[8,12]; a soft tissue mass[10,11]; an absence of calcification indicating a nonatherosclerotic origin of an aneurysm[6]; an adjacent abscess[10]; an atypical site of involvement of the aorta, such as thoracoabdominal[11]; and a lobulated cystic structure on an ultrasonogram.[13] The angiographic findings described are those of an eccentric saccular aneurysm arising from one wall of the vessel without other associated atherosclerotic disease.[12,13] The clinical rapidity of the development of an aneurysm and signs of an infection and predisposing conditions are necessary for the diagnosis.[12]

Scintigraphy using leukocytes labeled with indium 111 has been reported useful in one case of a clinically occult mycotic aneurysm of the thoracic aorta with subsequent computed tomography used to distinguish between an abscess and an aneurysm.[7] Ultrasonography has been previously reported to show an abdominal aortic aneurysm with a subsequent diagnosis by computed tomography.[10] These previous reports showed either gas collections within the aneurysm wall or an adjacent abscess with destruction of an adjacent vertebral body. One case report indicates a large retroperitoneal mass encasing the abdominal aorta found on CT that initially had been thought to be lymphoma but was actually a paravertebral abscess.[10] The CT scan in our case shows a ring of calcification that must represent the intima of the aorta. This has surrounding soft tissue that at the time was not clearly appreciated to be the lumen of an aneurysm. An ultrasonogram shows that this does represent the lumen of the aortic aneurysm. This is further confirmed by a digital subtraction angiogram. No gas in the soft tissue structures was seen, nor was there any bone destruction or adjacent abscess.

On histopathologic examination of the resected aneurysm in our case, there were atherosclerotic changes with foci of suppuration in the wall. Unfortunately, no organism could be cultured, probably owing to antibiotic therapy. We postulate that prosthetic valve endocarditis developed that was partially treated. Subsequently, a preexisting atherosclerotic aortic aneurysm became infected hematogenously. This initially produced vague abdominal pains and persistent fever but was diagnosed at angiography. A high degree of suspicion by a clinician leading to early angiography and subsequent surgical treatment can prevent a lethal end to this difficult diagnostic problem.

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